Prostate specific antigen level in seminal plasma of patients with oligospermia

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بسم الله الرحمن الرحيم

وقل ربِّ زدني علما

صدق الله العلي العظيم

Dedication

To my wife and sons

with

Love and respect

Dr. Haydar

I certify that this thesis was prepared under my supervision at the council of pathology in partial fulfillment of the requirement for the degree of fellowship of Iraqi Council for Medical Specialization in Chemical Pathology.

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List of contents

List of tables

List of figures

List of abbreviation

A	Absorbance
Α	Alpha
C°	Celsius degree
Cm	Centimeter
ELISA	Enzyme Linked Immuno Sorbent Assay
EIA	Enzyme Immuno Assay
FSH	Follicular Stimulating Hormone
HRP	Horse Radish Peroxidase
Hk	human glandular kallikrein
Kda	Kilodalton
LDH	Lactate Dehydrogenase
LH	Luteinizing Hormone
µl	Microliter
Ml	Milliliter
Mm	Millimeter
mg/ml	milligram per milliliter
ng/ml	nanogram per milliliter
Nm	Nanometer
P	Probability
PSA	Prostate Specific Antigen
R	Pearson correlation coefficient
r.p.m	revolution per minute
S	Serum
SD	Standard Deviation

Abstract

Background: The most common male infertility factors include azoospermia and oligospermia. Oligospermia refers to sperm densities of less than twenty million sperm per milliliter. In about 90% of cases of oligospermia, the reason is deficient sperm production. Unfortunately, in about 90% of cases the cause can not be identified and the condition is labeled idiopathic oligospermia.

The **aim** of present study is to show the possible changes in the level of PSA in serum and seminal plasma of oligospermic patients and compare to that of control group.

Methods: Between March 2004 and December 2004 , a total of 36 subjects were studied in the infertility clinic in Al-Elwiya's teaching hospital. Single measurements of PSA were carried out in the serum and seminal plasma samples obtained from 15 apparently healthy normal fertile male used as controls and 21oligospermic patients. The PSA was determined by ELISA.

Results: The mean age of cases group was 35.2±4.9 years while The mean age of control group was 34±5.2 years. The mean serum PSA concentration in cases and control groups was 2.5 ng/ml, showed no obvious or statistically significant differences between cases and controls. The mean seminal plasma PSA concentration of oligospermic patients was 2.1±0.8 mg/ml and 2.7±0.7 mg/ml among healthy controls which show obvious or statistically significant differences between cases and

controls($p<0.05$). The serum PSA concentration showed insignificant correlation with seminal plasma measurements among healthy control and cases groups. .

Conclusions: The seminal plasma PSA is reduced in oligospermic patients and this may be due to decrease secretion of PSA from prostatic cells into seminal fluid so it might be considered as parameter in male infertility diagnosis.

Chapter One

Introduction

1.1 Review:

Infertility is defined as the failure of a couple to achieve conception after one year of regular , unprotected intercourse[(1)].

ln the United States, it was estimated that as many as 15%of all couples have difficulty in conceiving a child . In about one third of the cases of infertility, it is male partner who is responsible, in another one third, it is the female who is responsible. Current estimates suggest about 6% of men between the ages of 15 and 50 are infertile [(1, 2)].

1.2 Male reproductive anatomy :

The male reproductive system as shown in (figure 1.1) include

1.2.1 Testes:

The testes have many convoluted seminiferous tubules which are site of sperm formation, the tubular wall is composed of developing germ cells; sertoli cells .The leydig cells, or interstitial cells, which lie in small connective tissue spaces between the tubules , are the cells that secrete testosterone [(1)].

1.2.2 Epididymis :

The epididymis consists of one long stretched tubule located on the dorsolateral side of the testicle and be functionally divided in to three main parts:

The caput (head) a main function is concentration of viable spermatozoa up to 100 fold by resorption of testicular fluid and of spermatozoa of lesser quality.

The corpus (body), post testicular maturation of sperm is an important feature of this section.

The cauda (tail), main feature is sperm storage, since some 70% of the spermatozoa are stored here in addition, spermatozoa of poor quality are resorbed [1,2].

1.2.3 Vas deferens :

A large thick- walled tubule, the two vas deferens one from each side course to the back of the urinary bladder base and become the ejaculatory ducts [1,3].

1.2.4 Prostate :

The prostate 3.5 – 4.0 cm in diameter and located around urethra below the bladder neck. It has multiglandular function. It has for example exclusive secretion of acid, zinc, citric acid and fibrinolytic enzymes responsible for the liquefaction of coagulated vesicular secretion. Prostate provides the first fraction of the ejaculate and constitutes 30% of its volume [4].

1.2.5 Seminal vesicle :

The seminal vesicle is formed by a paired convoluted tube of 5-6 cm in length and a diameter of 3-4 mm . It is located on the dorsal surface of the bladder. It is opening is located just below the ampulla of the ductus deferens . It has a multiglandular functions and exclusively secretes fructose and prostaglandins. Seminal vesicles contribute 70% to the ejaculate volume and delivers the last fraction of spermatozoa [1].

1.2.6 Cowper's gland :

Gland is a paired, pea-sized, typically mucous, compound tubular gland located directly below the prostate [1, 2] .

1.2.7 Glands of Littre :

Very small periuretheral glands, it is believed that the Cowper's gland and gland of Littre function to lubricate urethra [2,3].

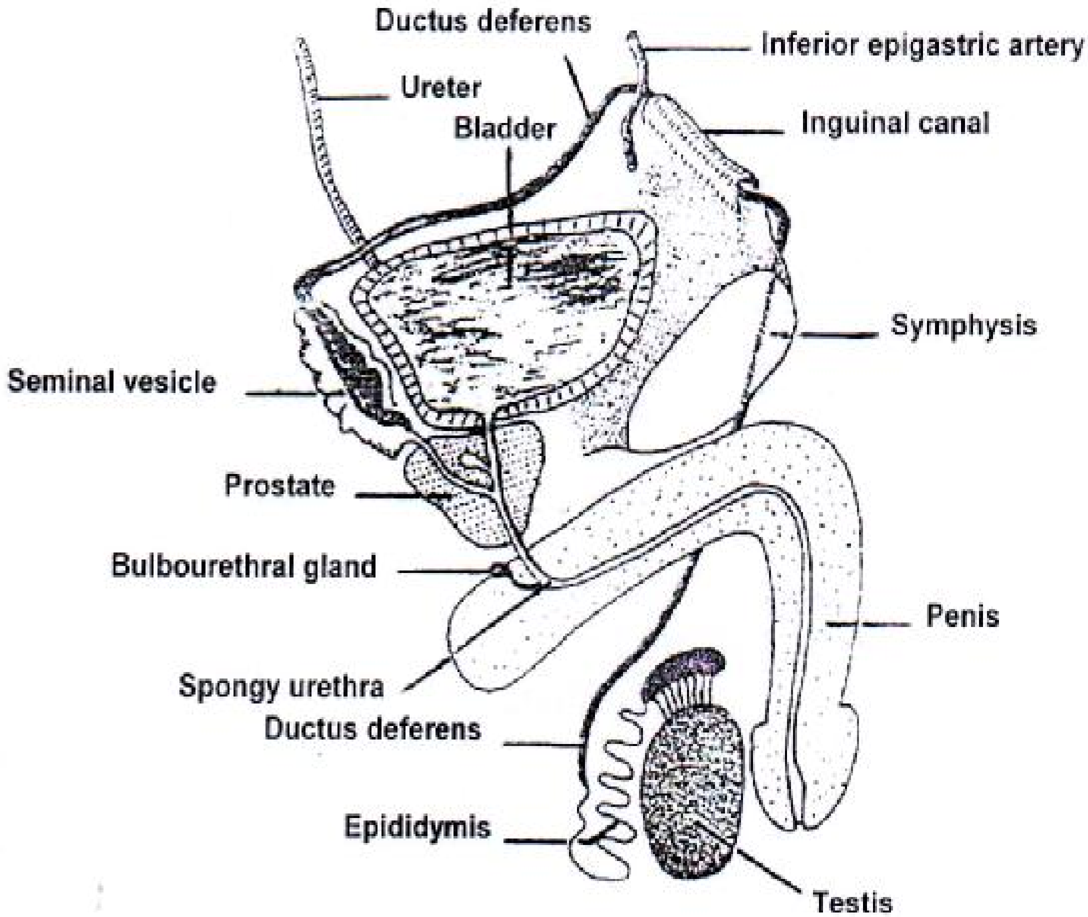


Figure 1.1: Male reproductive system [1]

1.3 Male reproductive physiology :

The human testis is an organ of dual functions:these are spermatogenesis (figure1.2) which occurs in the seminiferous tubules, and secretion of steroid hormone (androgens)by the leydig cells, which are present in the interstitial tissue. These testicular functions are intimately related, as testosterone synthesis is required not only for sperm production but also for the development of secondary sexual characteristics and normal sexual behavior [3].The anterior pituitary controls both these functions through secretion of gonadotropins, LH and FSH. In turn the anterior pituitary is regulated by many parts of brain via hypothalamic secretion of gonadotropin releasing hormone (GnRH) also known as lutenizing hormone releasing hormone (LHRH). This hypothalamic– pituitary – gonadal axis consist of closed loop feedback control mechanism directed for maintaining normal reproductive function [3, 4] (figure 1.3).

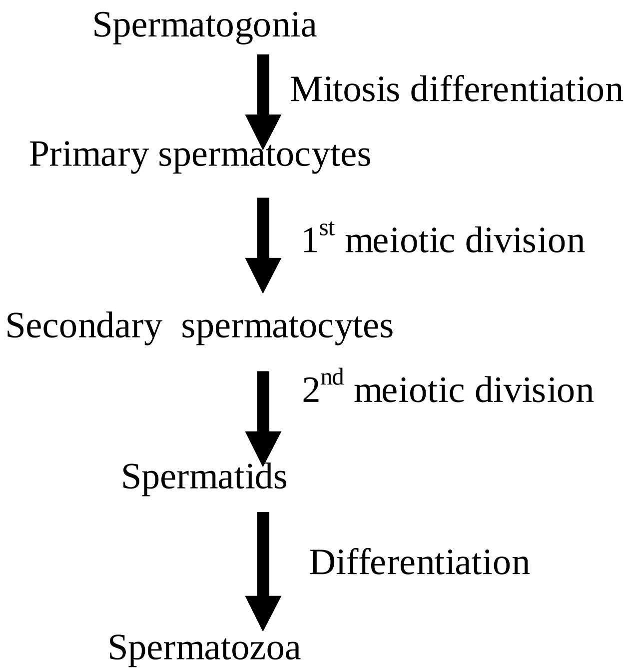


Figure 1.2: Diagram showing steps in spermatogenesis [3]

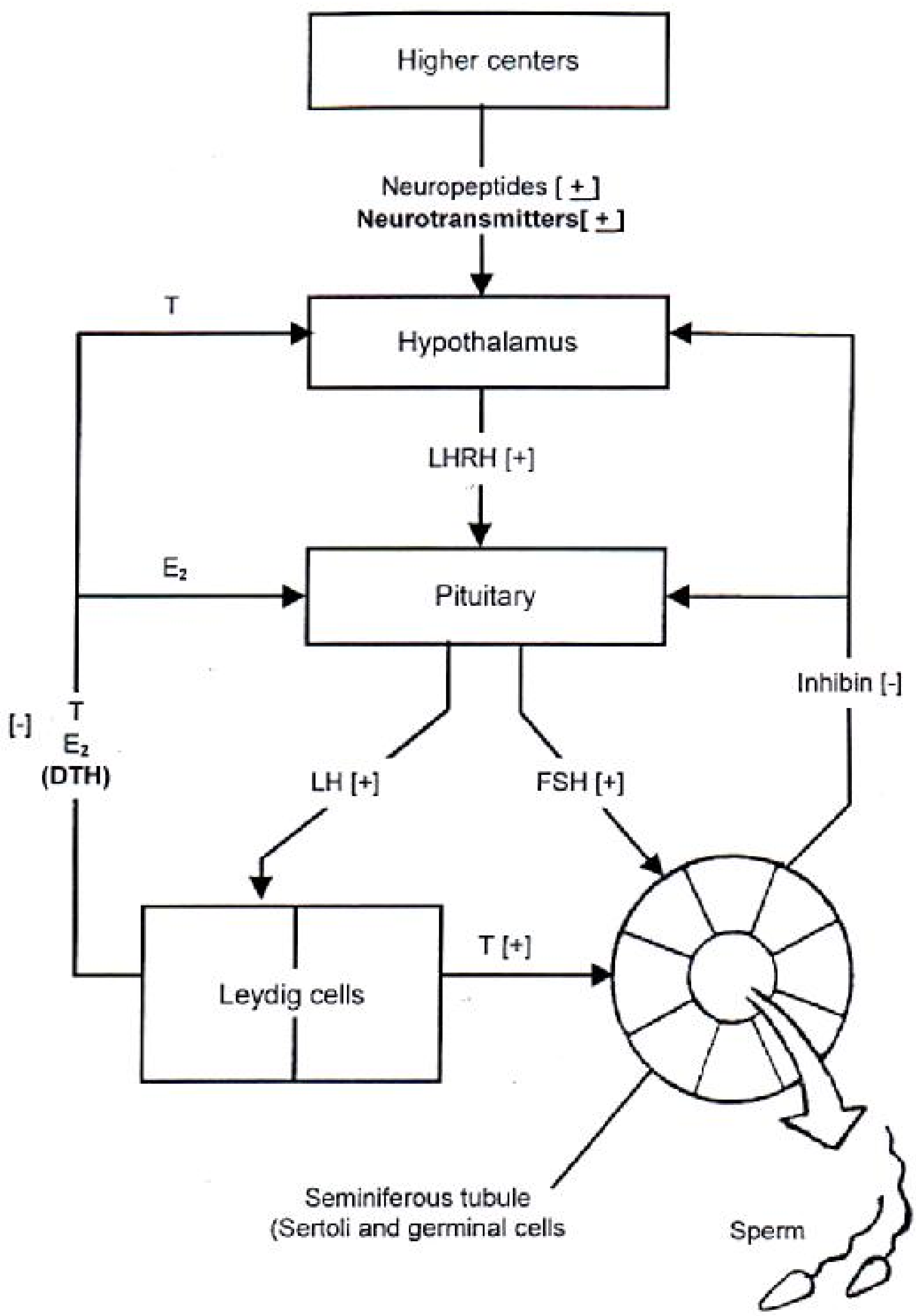


Figure 1.3: Hypothalamic-pituitary-gonadal axis. DHT = dihydrotestosterone: E2 = estradiol: FSH = follicle-stimulating hormone: LH = luteinizing hormone: LHRH = luteinizing hormone releasing hormone: T = testosterone: + = positive influence: - = negative influence (3)

1.4 Seminal plasma:

The seminal plasma is formed primarily from the secretions of the sex accessory tissues. Normal human ejaculate is approximately 3 ml and is composed of two components spermatozoa and seminal plasma. The spermatozoa which represent less than 1% of total ejaculate, are present in range of 100 million /ml[4]. The major contribution to the volume of the seminal plasma derives from the seminal vesicles. From prostate (1.5–2 ml), and from Cowper's gland (0.5 ml) and gland of Littre (0.1 – 0.2 ml). During ejaculation the secretions of these glands are released in a sequential manner. The first fraction of human ejaculate is rich in sperm and prostatic secretions. Fructose, which represents a major secretary product of seminal vesicles, is elevated in the later fraction of the ejaculate [5] .

In relation to other body fluid , the seminal plasma is unusual because of its very high concentrations of potassium, zinc, citric acid, fructose, phosphorylcholine, spermine, free aminoacids, diamine oxidase, beta glucuromidase , LDH, α–amylase, prostate specific antigen (PSA) and seminal proteinase[6].

1.5 Oligospermia:

The most common male infertility factors include azoospermia and oligospermia. Oligospermia refers to sperm densities of less than 20 million sperm per milliliter or a total count of less than 50 million sperm. Isolated oligospermia with normal movement and morphology parameters is uncommon. In sever oligospermia (less than 5–10 million per milliliter), hormone studies, specifically LH, FSH and testosterone level should be evaluated. If these levels are abnormal, a complete hormonal evaluation should be obtained [7].

In about 90% of cases of low sperm count, the reason is deficient sperm production. Unfortunately, in about 90% of cases, the cause for the decreased sperm formation can not be identified and the condition is labeled idiopathic oligospermia [8,9,10] .Other possible causes of oligospermia are shown in(table1.1)and (table1.2)

.

Table 1.1: Possible causes of oligospermia[11,12]

Increased scrotal temperature
Tight – fitting clothing and briefs
Varicocele are more common
Environmental
Increased pollution
Heavy metals (lead, mercury, arsenic…etc)
organic solvents
Pesticides
Hormonal
Hyperprolactinaemia
Hypothyroidism….. etc
Dietary
Increased saturated fat
Reduced intake of fruits, vegetable and whole grains
Reduced intake of dietary fiber
Increased exposure to synthetic oestrogen.

Table 1.2 : Causes of temporary oligospermia[13]

-Increased scrotal temperature
-Infection as flu, common cold
-Increased stress
-Lack of sleep
-Over use of alcohol, tobacco, and marijuana
-Many prescription drugs
-Exposure to radiations
-Exposure to solvent, pesticides, and toxins

1.6 Prostate Specific Antigen (PSA):

1.6.1Biochemical and physiological properties:

PSA also called P30 and gamma semino-protein [14,15]. PSA is a single chain glycoprotein that is 7% carbohydrate[14]. Functionally , PSA is an androgen regulated serine protease and member of the tissue kallikrien family of protease[14,16,17,18,19] . It is primarily produced by prostate ductal and acinar epithelium and is secreted into lumen, where its function is to cleave semenogelin I and II in the seminal coagulum[14,16,17,20]. PSA is normally found at much lower level in paraurethral and perianal glands, apocrine sweat glands, parotid glands, saliva, breast, thyroid and placenta [21, 22, 23].

PSA is synthesized with a seventeen – amino acid leader sequence (pre proPSA) that is cleaved contranslationally to generate an inactive 244 amino acids precursors protein (proPSA)[24] . Cleavage of the *N*- terminal seven amino acids from proPSA generate the active enzyme , which has five intrachain disulfide bonds, a single asparagine – linked oligosaccharide, and a mass of 33 kilodaltons(kda)[25,26,27] . This proPSA cleavage

normally occurs between the arginine at position 7 and isoleucine at position 8, with the isoleucine becoming the *N* – terminus of the mature active protein. This site can be readily digested by trypsin, but the major activating enzyme in vivo is human glandular kallikrien two (hK2), which has a trypsin like activity and is expressed predominantly by prostate secretary epithelium. PSA may also be activated by other prostate kallikriens, including prostase (hK4) [28].

Approximately 30% of PSA in seminal plasma is the intact proteolytically active enzyme and approximately 5%, is complexed with protein C inhibitor [29,30] . Additional forms are inactive because of internal cleavages (presumably by proteases in seminal fluid) between residues 85 and 86 , 145 and 146 or 182 and183[31] .

1.6.2 Molecular forms:

PSA exist in two major forms in blood circulation, PSA enters the peripheral blood and the majority (70% to 90%) is intact and circulates as an 80 to 90 kda complex with protease inhibitor α1–antichymotrypsin [14,32,33]. Minor amounts are complexed with other protease inhibitors including α2- microglobulin and α1–antitrypsin. PSA in peripheral blood that is catalytically inactive because of internal cleavages at residues 85 to 86, 145 to 146 or 182 to 183 does not form complexes with protease inhibitors or other proteins and circulate as free PSA comprising 10% to 30% of total PSA [14, 34] .

The metabolic clearance rate of PSA follows a two compartment model with initial half –lives of 1–2 and o.75 hour for free PSA and total PSA(hepatic clearance) and subsequent half –lives of 22 and 23 hours(renal clearance). Because of this relatively

long half – life, two to three weeks may be necessary for the serum PSA to return to base line levels after certain surgical procedures. Although the digital rectal examination has no clinically important effect on serum PSA levels in most patients, in some it may lead to two – fold elevation [14] .

1.6.3 Method of measurements:

Methods for detection of PSA include cross over electrophoresis, rocket immuno–electrophoresis, radial immuno – electrophoresis , radial immunodiffusion and ELISA .The extremely sensitive ELISA can detect PSA in concentration as low as approximately 4 ng/ml [14, 35].

1.6.4 Clinical applications:

PSA is useful in staging and monitoring of therapeutic responses and in evaluating the prognosis of the prostatic carcinoma. The studies have been shown that 98% of patients with stage D2 have a high PSA level [14,36] . There is a direct correlation between the volume of cancer and PSA level, with high PSA values (above 20 ng/ml) being indicative of clinically advanced cancer [14,37,38,39,40].

The sensitivity of PSA in the evaluation of disease progression in patients with advanced prostatic cancer is 86.7± 3.1% with specificity of 92.4± 4.1%, its accuracy is 89.2 ±1.7% [38] . PSA is significantly better than prostatic acid phosphatase, for detection of recurrence in patients under hormonal therapy. An increase in PSA can occur up to 12 months before detection by other methods such as ultrasound for example [38,41] .

Prostate specific antigen, new development beyond the prostate

Originally, it was thought that PSA was only produced by the cells of the prostate gland, however, non-prostatic PSA has been found in many female normal tissues, particularly breast tissues, parotid gland, endometrial tissue, breast milk, nipple aspirate, and amniotic fluid[42]. Also found in various tumor tissues including cancerous breast tissue, lung, ovarian and salivary gland tumors[43].

PSA levels vary during menstrual cycle with higher levels in women with excess androgen, idiopathic hirsutism and hirsutism of polycystic ovary syndrome [42, 44, 45].

Elevated PSA levels are seen in some breast and gynecologic cancers. A number of studies have indicated that these elevations are favorable prognostic factor in breast cancer [42, 46]. PSA expression was significantly associated with estrogen and progesterone receptor positively, young patient age, earlier disease stage, smaller tumor size, diploid tumors and tumor with low s-phase function [47]. PSA remained a significant independent prognostic factor after taking into account other clinical and pathological features [46].

Total immuno-reactive serum PSA is not correlated with patient diagnosis or tumor levels [48]. Serum free PSA may be more specific marker of breast tumors. Serum total PSA is found mainly bound to α1-antichymotripsin in normal women and in its free form in serum from women with both benign and malignant breast tumors [49, 50].

1.6.5 The normal range and cutoff value of PSA:

At present, the data suggests that a cut point of serum PSA is 4.0 ng/ml, however the normal range of serum PSA in males without prostatic disease rises with age, as a result of gland enlargement [51]. Some studies have shown that age- specific range for Blacks and Japanese differ from that of general population. So in addition to age, race specific PSA values have been suggested [40] (table 1.3). In seminal plasma, PSA is the major protein with concentration between 0.5-3.5 mg/ml [52].

Table(1.3): Age and race specific range of PSA values in general population ,Blacks, and Japanese[14,49]

Age(years)	Age and race specific S.PSA reference range (ng/ml)		
	General population	Blacks	Japanese
40-50	0-2.5	0-2.0	0-2.0
50-60	0-3.5	0-4.0	0-3.0
60-70	0-4.5	0-4.5	0-4.0
70-80	0-6.5	0-5.5	0-5.0

Aim of study:

Aim of present study was to evaluate the changes in the levels of serum and seminal plasma PSA of oligospermic patients as additional marker for diagnosis of male infertility and compare to that of control subjects.

Chapter Two

Subjects, Materials and Methods

2.1 Subjects :

Twenty one infertile male aged between 26 -43 years with a mean ±SD of (35.2±4.9 years), attended the infertility clinic in Al-Elwiya's Teaching Hospital. The patients were suffered from infertility of more than one year despite of regular coitus. Sperm counts were done for all patients .The inclusion criteria of cases group were the followings:

1-Normal hormonal profile.

2-No drug intake for last three months.

3-Normal genital organs by examination.

4-No rectal examination for last two weeks.

The control group consist of fifteen apparently healthy normal fertile male aged between 25 – 41 years with mean ± SD of (34.1 ± 5.2) years, having fathered a child before one year.

2.2 Materials:

Sample collection was conducted during a period starting from March 2004 to December 2004. A sample of blood and semen were obtained from each subject on the same day. Blood sample for PSA assay was used. Five ml of venous blood were collected into plain tube, allowed to clot and centrifuged at 2000 r.p.m for 10 minutes, aliquated and stored at -20 C^{o} until analyzed. Semen samples were collected in laboratory by masturbation after 3 to 4 days of sexual abstinence. The sperm count was determined by spermeter in

comparison of findings with WHO reference values. After the sample was allowed to liquefy in water bath at 37 C^o for 20-30 minutes. 10µl of semen without dilution was applied in spermeter chamber which contain 100 squares. The sperms were counted in 20 squares under microscope and the count calculated by multiplying the viewed sperm by million then divided by two. The rest of semen sample was centrifuged at 2000 r.p.m for 15 minutes. The supernatant aliquated and stored at – 20 C^o.

Seminal plasma (after serial dilution) and serum PSA was determined by enzyme immuno- assay, using PSA enzyme immunoassay test Kit provided by Biocheck- United States.

2.2.1 Principle of procedures:

The PSA ELISA test is based on the principle of a solid phase enzyme – linked immunosorbent assay. The assay system utilizes a rabbit anti – PSA antibody directed against intact PSA for solid phase immobilization (on the microtiter wells). A monoclonal anti – PSA antibody conjugated to horseradish peroxidase (HRP) is in the antibody – enzyme conjugate solution. The test sample is allowed to react first with the immobilized rabbit antibody at room temperature for 60 minutes. The wells are washed to remove any unbound antigen. The monoclonal anti –PSA – HRP conjugate is then reacted with the immobilized antigen for 60 minutes at room temperature resulting in the PSA molecules being sandwiched between the solid phase and enzyme – linked antibodies . The wells are washed with water to remove unbound – labeled antibodies. A solution of TMB reagent is added and incubated at room temperature for 20 minutes, resulting in the development of a blue color. The color development is stopped with the addition of stop solution changing the color to yellow. The concentration of PSA is directly proportional to the color

intensity of the test sample. Absorbance is measured by EIA reader (Biorad 550) in central health laboratory at 450 nm.

2.2.1 Calculation of results:

1-Calculate the average absorbance values (absorbance 450nm) for each set of reference standards , control and samples.

2-Construct a standard curve by plotting the mean absorbance obtained for each reference standard against its concentration in ng/ml on linear graph , with absorbance on the vertical(y) axis and concentration on the horizontal (x) axis (figure 2.1) .

3-Using the mean absorbance value for each sample , determine the corresponding concentration of PSA in ng/ml from the standard curve.

2.3 Statistical methods:

1-The significance of difference between mean values was estimated by independent sample t – test.

2-Pearson correlation coefficient (r) was used to test the relation between the parameters.

P value less than the 0.05 level of significance was considered statistically significant.

The analysis of data was a accomplished by using the statistical package, for social sciences SPSS/ PC+, the statistical package for IBM PC.

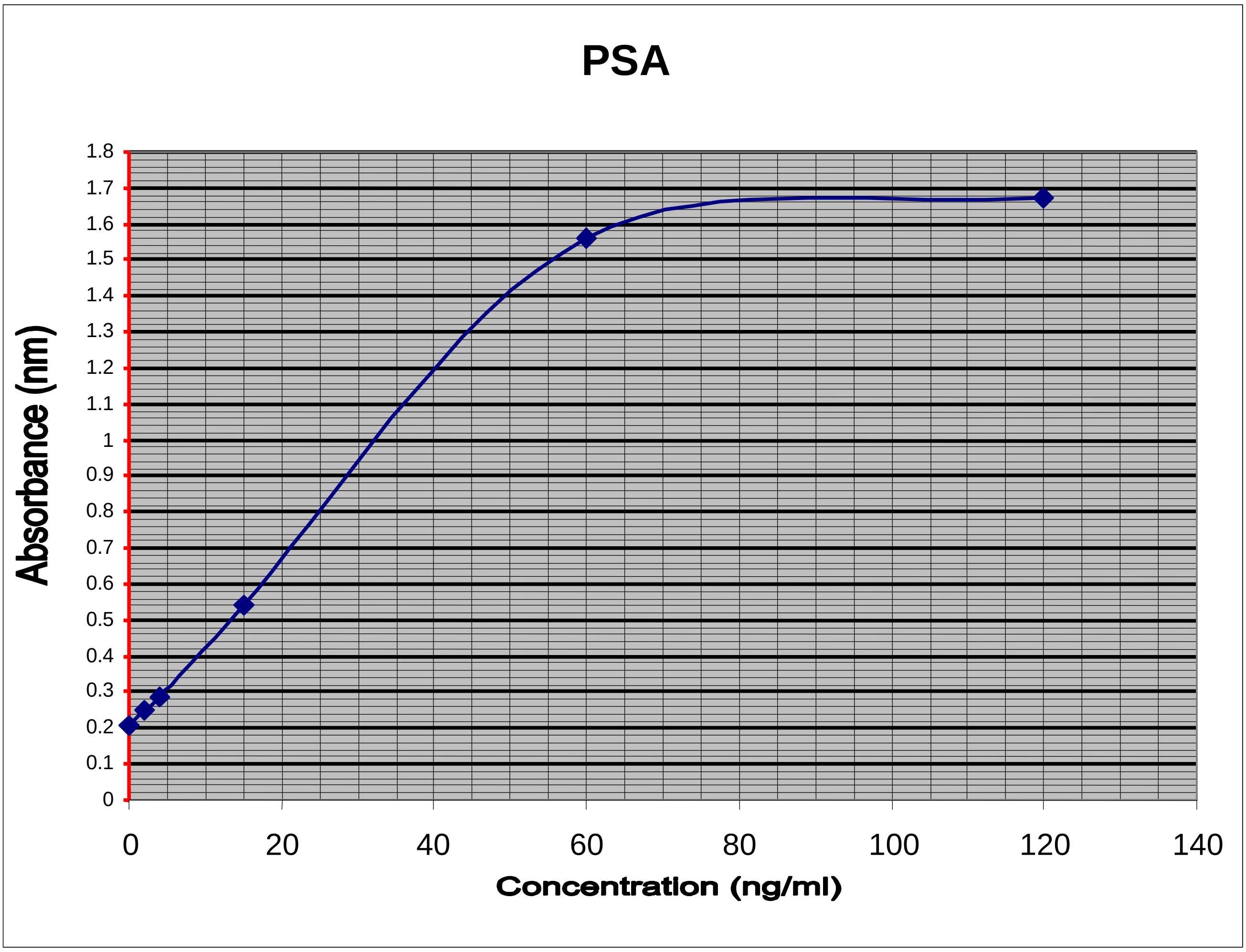


Figure 2.1 : Standard curve of PSA

Chapter Three

Results and Discussion

The results presented in this chapter were based on the analysis of data on 21 infertile males with oligospermia (cases group) and 15 healthy control subjects.

3.1 Description of study samples:

The age of infertile group ranges between 26-43 years with a mean ± SD of 35.2 ± 4.9 years, which was not significantly different from that of the control group (34.1 ± 5.2 years), (table3.1) i.e. the procedure of group matching on age in selecting the healthy control group was effective in excluding age as a possible confounder in explaining any case-control difference in outcome variables (like PSA concentration).

As shown in (table3.2) and (figure3.1), the sperm concentration of cases group with oligospermia ranged between 1-19 million/ml with a mean ± SD of 11.4 ± 5.2 million/ml, which is obviously and significantly lower (as expected) than healthy control group (91.7 ± 17 million/ml).

A great attention has been directed to the chemical components of human seminal plasma which could be measured by biochemical methods in ejaculate. There was an Iraqi study that measure zinc , acid phosphatase , fructose, L-carnitine and α-glucosidase in seminal plasma of obstructive and non obstructive azoospermic patients [53]. PSA is the major protein in seminal plasma [52]. In this study, PSA in the serum

and seminal plasma of patients with oligospermia was estimated.

Table 3. 1: Case-control difference in mean age

	Control group (n=15)	Infertile group (Oligospermia) (n=21)	P (t-test)
Age in years			0.51[NS]
Median	35	36	
Mean	34.1	35.2	
SD	5.2	4.9	
SE	1.35	1.07	

Table 3. 2: Case-control difference in mean sperm concentration

	Control group (n=15)	Infertile group (Oligospermia) (n=21)	P (t-test)
Sperm concentration (x million / ml)			<0.001
Median	90	12	
Mean	91.7	11.4	
SD	17	5.2	
SE	4.38	1.13	

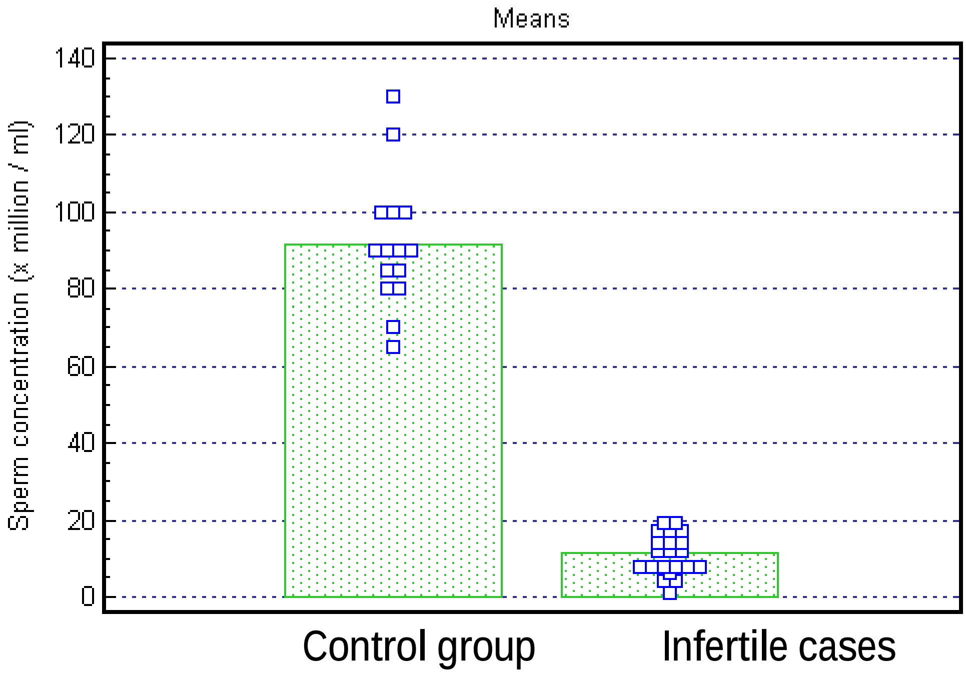


Figure 3.5:Dot diagram showing the case (infertile) control difference in means of sperm concentrations

3.2 Serum and semen PSA concentration:

Case-control difference:

As shown in (table 3.3) and (figure 3.2), the mean of serum PSA concentration of control group was 2.5±0.7 ng/ml, similarly the mean of serum PSA concentration in oligospermic patients was 2.5±0.9 ng/ml. The level of serum PSA were in the normal range in both oligospermic and control groups[14]. The mean serum PSA showed no obvious or statistically significant difference between oligospermic patients and controls. The mean of seminal plasma PSA concentration was 2.1±0.8 mg/ml in oligospermic patients and 2.7±0.7 mg/ml in healthy controls. The mean seminal plasma PSA was significantly lower among cases with oligospermia than among healthy controls ($P<0.05$)(table 3.3). The reason might be due to impaired secretion of PSA in oligospermic patients from prostatic cells into seminal fluid .The function of PSA in seminal fluid is cleavage of semenogelin I and II in the seminal coagulum[14,16,17,20]. So low PSA might leading to infertility as clotted semen does not liquefy so the sperm trapped in the clot and will unable to move toward the ovarian follicle. PSA enters the peripheral blood from prostatic secretion [14,32,33]. The serum PSA levels of oligospermic patients in this study remain within normal range, the reason may be due to other sources which might contribute to the serum PSA as thyroid , parotid gland, apocrine sweat glands and perianal glands [18,19,20].

Table 3-3: Case-control difference in mean serum and seminal plasma PSA

	Control group (n=15)	Infertile group (Oligospermia) (n=21)	P (t-test)
Serum PSA(ng/ml)			0.99 *[NS]*
Median	2.5	2.5	
Mean	2.5	2.5	
SD	0.7	0.9	
SE	0.18	0.19	
Seminal plasma PSA (mg/ml)			0.02
Median	2.8	1.9	
Mean	2.7	2.1	
SD	0.7	0.8	
SE	0.18	0.17	

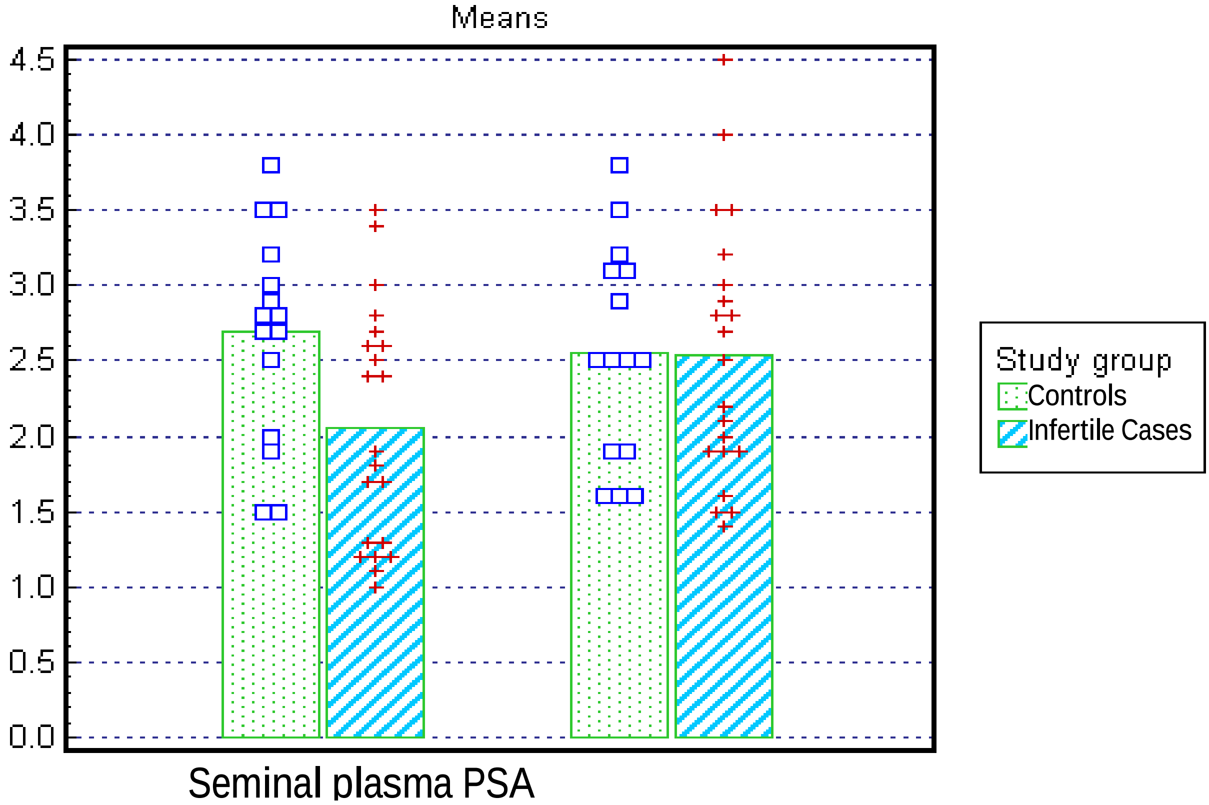


Figure 3-6: Dot diagram showing the case (infertile) control difference in mean and distribution of values of serum and seminal plasma PSA concentration

3.3 Linear correlation between serum and seminal plasma PSA measurements

As shown in (figure3.3) and (figure3.4), the serum PSA showed a weak and statistically insignificant positive linear correlation with seminal plasma PSA measurements among healthy controls and of oligospermic group (r=0.25, r=0.35 respectively).

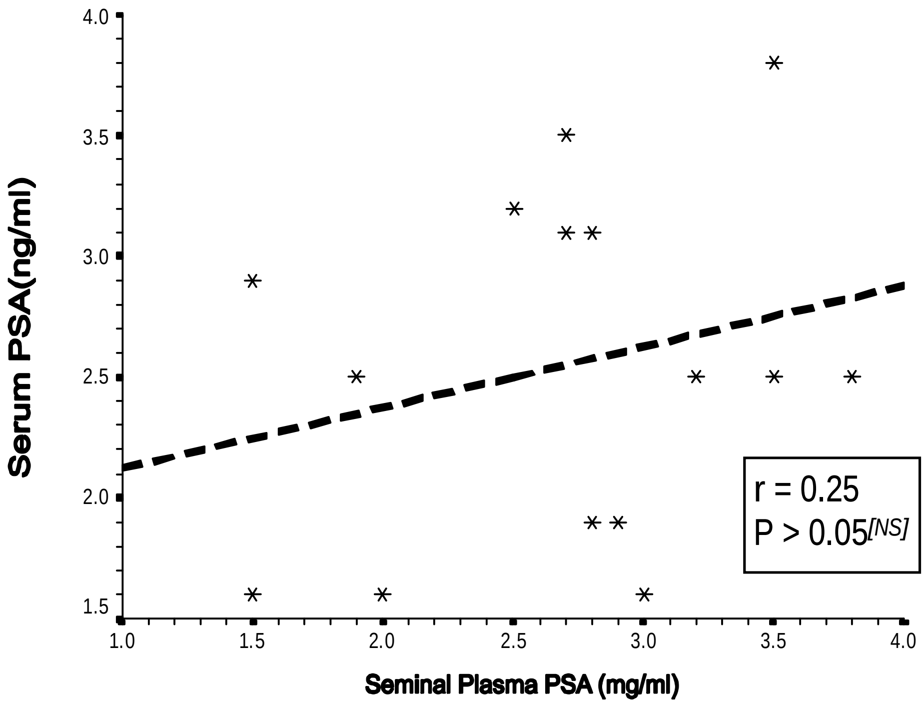


Figure 7.3: Scatter diagram (with fitted regression line) showing the linear correlation between seminal plasma and serum PSA among control group

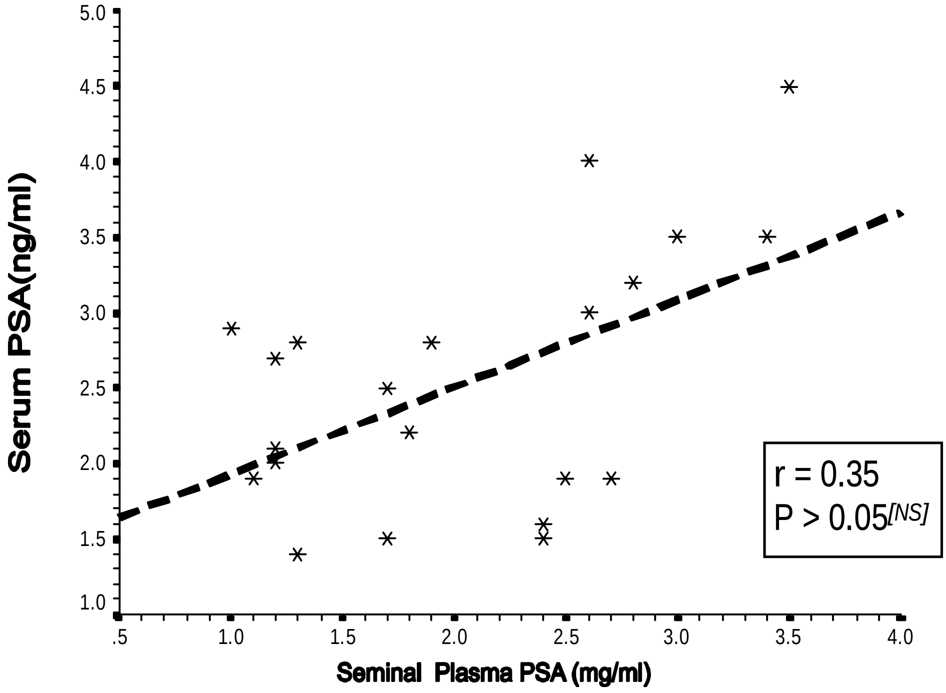


Figure 3.8: Scatter diagram (with fitted regression line) showing the linear correlation between seminal plasma and serum PSA among oligospermic group

3.4 Linear correlation of seminal plasma PSA with sperm concentration:

As shown in (figure3.5) and (figure3.6), among healthy controls,the sperm concentration showed no obvious or statistically significant linear correlation with seminal plasma PSA concentration. Among cases with oligospermia the same parameters showed a weak and statistically insignificant positive (direct) linear correlation.

Although seminal plasma PSA concentration was significantly lower among cases with oligospermia, its magnitude do not correlate with sperm concentration.

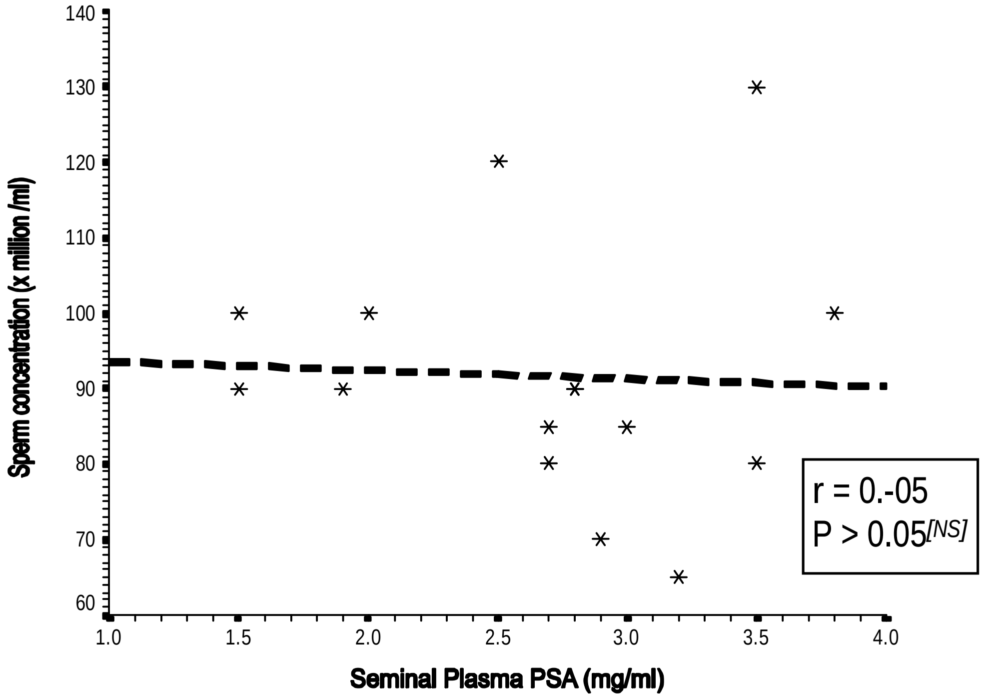


Figure 3.9: Scatter diagram (with fitted regression line) showing the linear correlation between seminal plasma PSA and sperm concentration among control group

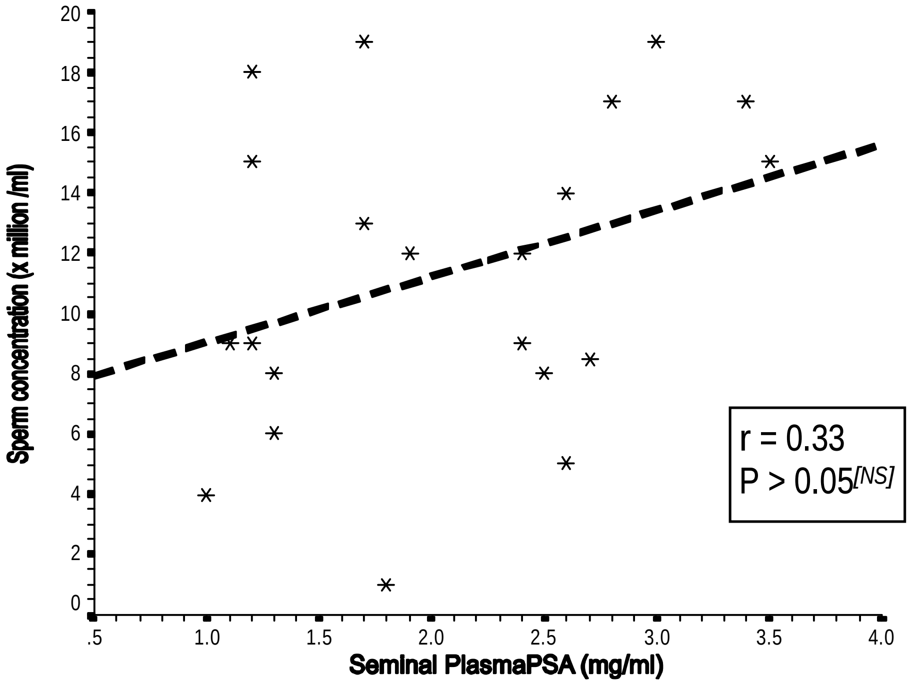


Figure 3.10: Scatter diagram (with fitted regression line) showing the linear correlation between seminal plasma PSA and sperm concentration among oligospermic group

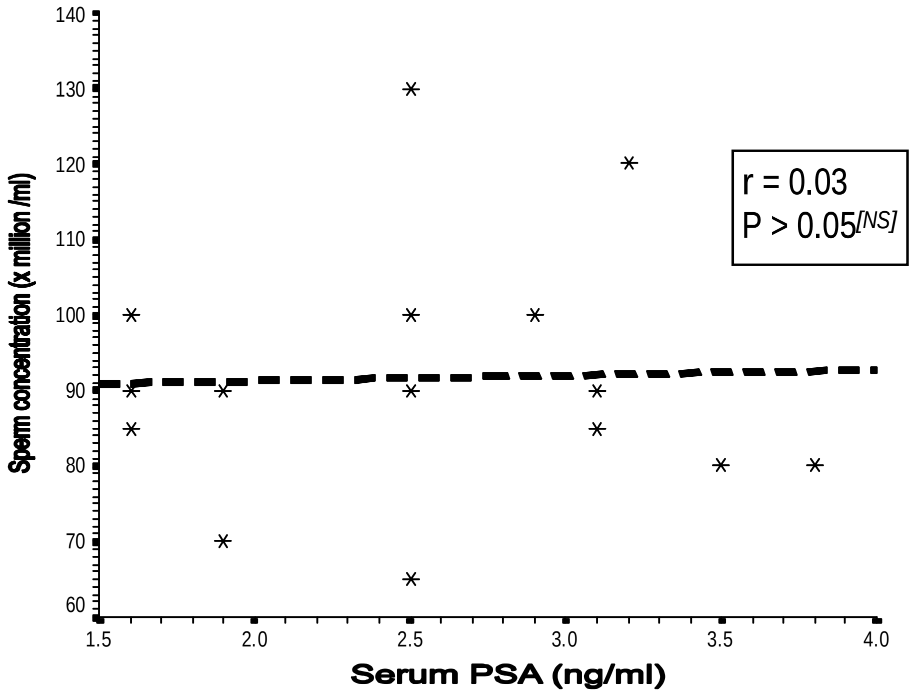


Figure 3.11: Scatter diagram (with fitted regression line) showing the linear correlation between serum PSA and sperm concentration among control group

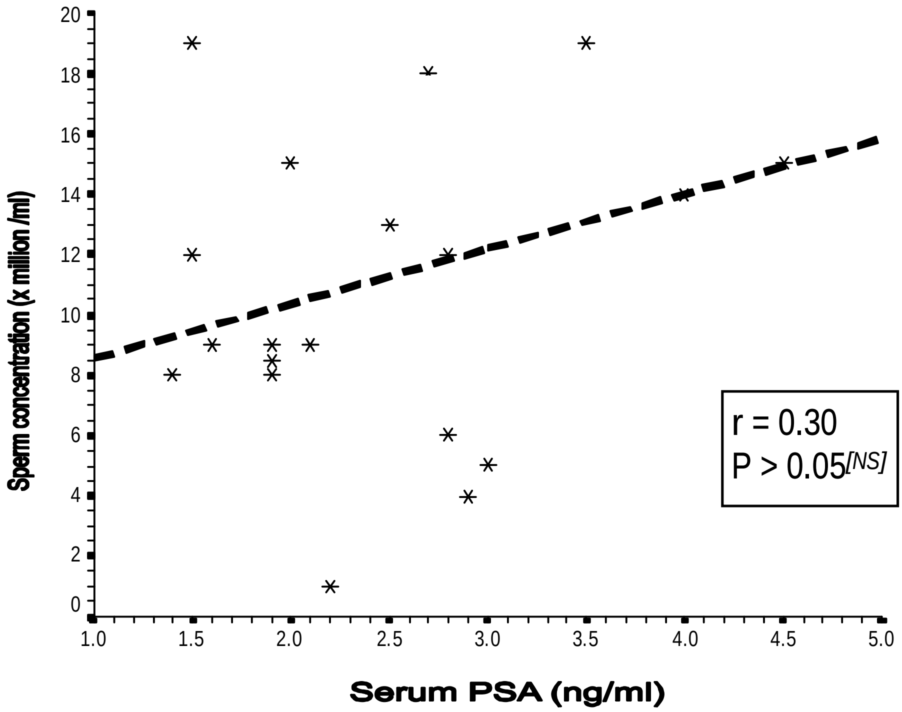


Figure 3.12:Scatter diagram (with fitted regression line) showing the linear correlation between serum PSA and sperm concentration among oligospermic group

Conclusion

On the basis of the results, this preliminary study demonstrated that the level of PSA in seminal plasma was reduced in patients with oligospermia that might give a clue in changing the modality of treatment in such patients.

Recommendation

Seminal plasma PSA could be introduced with other biochemical marker such as L-carnitine and fructose that used to assess male infertility.

Suggestion for future studies

Further studies are suggested to show the relation between seminal plasma PSA and viscosity in patients with oligospermia.

Chapter Four

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الخلاصة

الخلفية : من اهم عوامل عقم الذكور هي عدم وجود المني والصلْد (قلة المني) . يطلق مصطلح الصلد عندما تكون كثافة الحيامن اقل من عشرين مليون في المليلتر الواحد ويعود السبب في ٩٠% من حالات الصلد الى عدم انتاج الحيامن ، وفي ٩٠% من تلك الحالات يكون السبب مجهولاً وتسمى حالة الصلد عندئذٍ بالصلد المجهول السبب .
الغرض من اجراء هذه الدراسة هو لمعرفة التغير الذي قد يحصل في مستوى مستضد البروستات الخاص في مصل الدم والبلازما المنوي لمرضى الصلد ومقارنتهم بالاشخاص الطبيعيين .

الطريقة : بين آذار وكانون الأول من عام ٢٠٠٤ تم دراسة ٣٦ شخصاً في عيادة مرضى العقم في مستشفى العلوية التعليمي حيث تم قياس مستوى مستضد البروستات الخاص في الامصال والبلازما المنوي لخمسة عشر شخصاً طبيعياً وواحد وعشرين مريضاً مصاباً بالصلد . استخدمت طريقة الاليزا لقياس تركيز مستضد البروستات الخاص .

النتائج : كان معدل اعمار مرضى الصلد هو ٣٥ ± ٤.٩ سنة و ٣٤ ± ٥.٢ سنة بالنسبة للاشخاص الطبيعيين . كان معدل مستضد البروستات الخاص في امصال الاشخاص الطبيعيين ومرضى الصلد هو ٢.٥ نانو غرام في المليلتر الواحد ووجد بأنه لا يوجد هناك اي اختلاف بين معدل الاثنين في حين بلغ معدل مستضد البروستات الخاص في البلازما المنوي للاشخاص الطبيعيين ٢.٧ ملغم / مل ± ٠.٧ و ٢.١ ملغم / مل ± ٠.٨ بالنسبة لمرضى الصلد ووجد بأن هناك اختلاف بين معدل الاثنين (مستوى الاحتمالية اقل من ٠.٠٥) ووجد بأن تركيز مستضد البروستات الخاص لايحمل اية علاقة لمؤشر البلازما المنوي للاشخاص الطبيعيين والمرضى.

الاستنتاج : ينخفض مستوى مستضد البروستات الخاص في البلازما المنوي لمرضى الصلد وقد يكون السبب في ذلك الى انخفاض افرازه من خلايا البروستات لذلك من الممكن استخدامه كعلامة تشخيصية لهؤلاء المرضى .

مستوى مستضد البروستات الخاص في البلازما المنوي لمرضى الصلد (قلة الحيامن)

رسالة مقدمة إلى
المجلس العراقي للاختصاصات الطبية

وهي جزء من متطلبات درجة البورد
في الكيمياء الباثولوجية

من قبل الدكتور
حيدر هاشم الشلاه

بأشراف
الأستاذة المساعدة الدكتورة أنسام البياتي

صفر ١٤٢٦ هـ   نيسان
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